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EAR, NOSE AND THROAT

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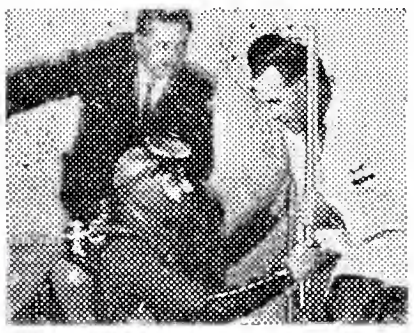

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Compassion, Courtesy, Concern for Citizen and Community Compels Clinicians to Co-opt Communication as a Constant Companion

The establishment of the University of Toronto Medical Journal 50 years ago tacitly acknowledged the importance of communication, communication with the profession, the public and the patient as part of medical education. Communication is not merely a superficial and sometimes confusing display of information but an earnest attempt to insure that meaning and significance are clearly presented so that they may be comprehended.

At the time the Journal began publishing those who entered Medicine were expected to have some familiarity with the classics. An exposure to Latin or Greek, English and History were considered the hallmarks of the educated man. It is an impression, hard to confirm, that this background did indeed enhance the individual's ability to communicate particularly through the medium of the written word.

Present requirements emphasize a scientific training. To-day, assessment of student performance is by multiple choice examination, a method providing a more objective assessment of a student's fund of knowledge than an essay type examination but the essay type examination required him to demonstrate not only his knowledge but his ability to communicate it. We may have sacrificed an essential art on the altar of computerized marking.

A large percentage of all articles presented for publication to medical scientific journals are so poorly constructed, the language so garbled and the charts and illustrations so confusing as to render them almost incomprehensible. Numbers of interesting scientific communications are rejected by editors for lack of clarity in presentation. One of the largest medical journals on this continent for these reasons rejects outright 85% of the manuscripts submitted for publication. The medium has failed to convey the message. This circumstance has spawned a new profession; consultant in biomedical communications who assist in the preparation of scientific articles, lectures, movies and television, usually for a respectable fee. Indeed, courses in scientific communication are now presented at many of the national and international meetings of major professional societies such as the Royal College of Physicians and Surgeons of Canada and the College of Surgeons of U.S.A.

A pioneer project of this nature in Canada was the development of a Department of Medical Communications at The Hospital for Sick Children in Toronto. An elective in biomedical communications has been offered to undergraduates but there have been no applicants for the programme to date.

It has been suggested that audio visual devices are likely to largely replace the printed word as the means of communication of the future. Even if this is true, all such devices to be successful, depend on the prior preparation of a lucid, logically constructed script.

A clinician does not communicate with his peers alone. He must treat his patients, with compassion and courtesy; he must express himself and be active in the concerns of his community. One reason the profession is in its present state of disarray stems from a failure to co-opt the art of communication.

It is a matter of pride and satisfaction that through the years the Medical Journal has provided a stimulus to students to learn to express their interests in writing and a forum to present the results of their efforts. I hope that an increasing number of undergraduates will avail themselves of this opportunity to communicate.

A. L. CHUTE, O.B.E., M.A., M.D., Ph.D., F.R.C.P.(C)
Dean of the Faculty of Medicine
University of Toronto

Guest Editorial

DR. MICHAEL HAWKE is a graduate of University of Toronto (1965). As an undergraduate, his interest in clinical research led to a B.Sc. (Med.). He completed his residency in otolaryngology in Toronto which included work in otolaryngic pathology at the Armed Forces Institute of Pathology, Washington, D.C. He obtained his diploma and his certificate in ENT in 1970. Since then, he has worked actively in the Temporal Bone Histopathology Laboratory and as a consultant in ENT.

In 1971 Dr. Hawke was given cross-appointments in the Dept. of Otolaryngology and Dept. of Pathology and in 1972 he became assistant professor in the Dept. of Otolaryngology. Dr. Hawke is on staff at Toronto General Hospital and St. Joseph's Hospital.

Otorhinolaryngology, more commonly known as Otolaryngology, Ear, Nose and Throat, or E.N.T., is one of the oldest, and yet most misunderstood specialties. This specialty, unfortunately, is construed by many people to involve predominantly the surgical operation of adenotonsillectomy.

The earliest physician whose name is recorded as dealing with a specific otorhinological condition was the Egyptian Sekhet'enanch, who was the Medical Attendant of Pharoah Sahura, of the Fifth Dynasty (around 3500 B.C.). A limestone stele with a drawing of the physician and his wife, bearing the inscription, "He healed the King's nostrils" was found in Sahura's tomb.

The earliest medical writings found in the Papyrus Ebens, which was probably written around 1550 B.C. listed a large number of empiric remedies including remedies for deafness and aural discharge. For example, "For an ear that hears badly — red lead, and resin; grind to a powder, rub in fresh olive oil, and apply to the ear", and, "For an ear that is suppurating, olive oil, frankincense and sea salt, syringed into the ear".

The oldest known surgical treatise, the Edwin Smith Papyrus (1600 B.C.) includes an excellent description of the treatment of a fractured nose: "Every worm of blood is to be removed, the displaced bone is to be forced back, and the nostrils packed with strips of linen saturated with grease and honey".

Over the intervening years, the specialties of otology, rhinology and laryngology were developed as the knowledge of the anatomy of these areas and disease processes affecting them became known. However, it was not until the dawn of the 20th century that these three specialties became united under the comprehensive term of Oto-laryngology.

Otolaryngology today is both a medical and a surgical specialty. In Otology with the advent of the antibiotic era, the emphasis has shifted from the management of suppurative disease to the preservation of hearing and reconstruction of the middle ear and tympanic membrane. The use of the operating microscope and modern micro-surgical instruments has opened up a whole new field of micro-oto-surgical procedures, e.g. tympanoplasty, stapedectomy for otosclerosis, facial nerve decompression, etc.

The modern otolaryngologist is concerned with the prevention of hearing loss in such areas as maternal rubella, noise induced hearing loss (acoustic trauma), and the proliferation of new ototoxic drugs (e.g. the *Streptomyces* derivatives, ethacrynic acid, etc.). Special programmes for the early detection and education of the hearing handicapped child are being developed at the Hospital for Sick Children.

Otoneurology has recently become a highly developed branch of Otology with the establishment of five special vestibular testing units in the Toronto area. A Temporal Bone Histopathology Laboratory has been jointly developed by the Departments of Pathology and Otolaryngology to study the microscopic anatomy and pathology of the temporal bone in order to better understand those disease processes which affect the ear. The Ontario Temporal Bone Bank programme has been established in order to provide a mechanism by which patients who have or who have had ear disease may will their temporal bones for microscopic study.

In rhinology, the emphasis has shifted from the management of chronic inflammatory disease to reconstructive procedures of the internal and external nose, the investigation of nasal airflow and nasal allergy, and regional nasal plastic surgery (cosmetic rhinoplasty).

In laryngology, attention is being directed at the recently recognized complications of prolonged intubation and tracheotomy (glottic and subglottic stenosis). The application of the operating microscope and special micro-surgical instruments has paved the way for more delicate and precise intralaryngeal surgery.

Carcinoma of the larynx is still a serious and not uncommon problem which is now being evaluated and managed by a team approach combining radiotherapist and otolaryngologist. There is a renewed emphasis on conservation surgery which enables the patient to retain both voice and trans-laryngeal respiration.

The invention of the flexible fiber optic endoscopes, bronchoscope, laryngoscope, esophogascope and gastroscope has greatly improved the ease and accuracy of peroral endoscopy.

The more common diseases affecting the ear, nose and throat (e.g. acute upper respiratory infections, nasal airway obstruction, sinusitis, epistaxis, acute otitis externa, otitis media, vertigo, disorders of hearing and dysphagia) account for approximately 20% of the patients seen by the family practitioner in his office.

Despite this high incidence, it is regrettable that the amount of time devoted to the study of this area has been relegated by the Curriculum Committee to a period of time of approximately one week during Period II and two weeks during Period III.

This short period of time is inadequate to obtain the proper level of skill in using the head mirror, nasal speculum, nasopharyngeal and laryngeal mirrors to develop the basic skills needed to properly manage common clinical conditions.

The Department of Otolaryngology of the University of Toronto has available several electives for undergraduate students who would like to examine some of these areas in greater detail. The Conacher Laryngeal Library and the Atkinson Otological Library contain a wealth of educational and self-instructional material, including reprints, tape-slide presentations, movies, television tapes, and teaching models which can be made available to those interested, upon request.

Editor's Note

This is the first issue of Volume 50 of the *Journal* making it, to the best of our knowledge, the oldest publication of its kind in North America. It first appeared in November 1923 with two main objectives in mind: first, to convey information of academic and practical interest to undergraduates which they might not otherwise obtain, and second, to provide medical students a chance to develop skills of writing and research which would be useful to them after graduation. The successful attainment of these goals obviously requires people who write articles and people who read them, so I would encourage every medical student to read the *Journal* and those who feel capable to submit articles for publication. In addition I would like to ask any of our readers, graduates or undergraduates, who have any thoughts or criticisms about the *Journal* to send them along to us. With continued broad-based suport we can approach the next 50 years with every hope of further success.

D.W.

Epistaxis: Control And Management

Anthony F. Jahn, B.Sc., 7T4

Persistent epistaxis is a condition that plagues both the patient and his physician. Bleeding from the nose may have many causes, both local and systemic, and this article will outline a rational approach to dealing with this common problem.

Before considering actual treatment, we should review the vascular anatomy of the nose, since effective control must be based on familiarity with the distribution of arterial supply. Lucid descriptions of nasal vasculature are rare, and the pathways of blood vessels are most easily visualized with the aid of the dry skull.

It is easiest to consider the arteries of the nose as approaching the nasal cavity from four directions: from above, from the front, from below and from behind. *From above*, terminal branches of the anterior and posterior ethmoidal arteries pass medially and down from the bony orbit to supply the highest reaches of the nasal vault. These vessels are branches of the opthalmic artery which is derived from the internal carotid artery. Otherwise, the nose is supplied by branches of the external carotid exclusively. *From the front*, terminal septal branches of the superior labial artery pass up and into the nose, next to the anterior nasal spine. The labial artery is derived from the facial (external maxillary) artery, a branch of the external carotid. The rest of the nose is supplied by the maxillary (internal maxillary) artery, a terminal branch of the external carotid. Due to its great importance in epistaxis, this vessel has earned the sobriquet "the rhinologist's artery". The maxillary artery ascends in the pterygopalatine fossa and gives rise to two branches: the greater palatine artery and the sphenopalatine artery. The greater palatine artery descends through the greater palatine canal and enters the oral cavity through the greater palatine foramen, medial to the third maxillary molar. The artery courses forward and medially along the palato-alveolar junction and passes up the incisive foramen in the midline. It thus enters the nose *from below* to supply the anterior septum. The sphenopalatine artery passes medially through the sphenopalatine foramen to enter the nose *from behind*, just above and behind the middle turbinate shelf. It gives off a smaller branch, the descending palatine artery, which meets the ascending palatine branch of the facial artery to supply the posterior margin of the choanae as well as the palatine tonsils. If a stiff wire is passed through the pterygomaxillary fossa of the dry specimen, the pathways of these vessels are easily demonstrated.

To summarize (from the nasal point of view), the lateral wall of each nasal cavity is supplied by: the anterior and posterior ethmoidal arteries (to the apex of the nasal vault and superior turbinate), greater palatine artery (to the anterior region below the inferior turbinate) and sphenopalatine artery via its posterior lateral nasal branches (to the rest of the lateral nasal wall). Each side of the septum is supplied by the sphenopalatine artery via its posterior nasal septal branch (to the posterior septum), the posterior and anterior ethmoidal arteries (to the superior septum) and by the terminal anastamosis of sphenopalatine artery, greater palatine artery and superior labial artery (to the anterior septum). These form a richly vascularized network and the anterior inferior septum, called Kiesselbach's plexus. The region is known as Little's area.

The primary sites of epistaxis can now be delineated anatomically. Nosebleeds are grouped roughly as anterior or posterior. Ninety percent of nosebleeds are anterior and originate in Little's area. Anterior epistaxis may occasionally issue from high in the anterior nasal vault which is anterior ethmoidal territory. The commonest site of posterior epistaxis is just postero-superior to the posterior end of the middle turbinate, where the sphenopalatine artery enters the nose. Another area of clinical import is the venous plexus underneath the posterior end of the inferior turbinate[1]. While bleeding may arise anywhere in this highly vascular organ, these four sites account for the vast majority of nasal haemorrhages.

Control of Nosebleeds

Epistaxis is the single result of multiple causes and since attention is first directed toward hemostasis, we will consider immediate treatment first. Longer term therapy of course is modified by history and a thorough physical examination, and this aspect of total management will be discussed later.

Historical accounts of epistaxis management range from the exotic to the ridiculous and include snake venom, salted pork strips inserted into the nostril, tampons made of water-filled sheep oesophagus, moxibustion, etc.[2, 16]. They reflect our basic inability to reliably and predictably control the bleeding. Some basic assumptions can be made upon approaching the patient with epistaxis. We should assume that bleeding originates from one side of the nose and that there is a single bleeding site. We may also assume the nosebleed to probably originate anteriorly, since statistically the great majority do. This assumption is justifiable especially in the young or where there is trauma to the external nose only. The assumption is modified in older patients who demonstrate increasing propensity for posterior nosebleeds[3]. If a patient spits blood, this may be suggestive of a bleeding site near the nasopharynx, but a clot blocking the nasal passage causing a backup of blood into the throat may also give this picture. Particularly severe epistaxis may pour blood into the throat regardless of actual site of bleeding. With an occluded nasal passage, as with deeply posterior bleeding sites, blood may course around the posterior edge of the vomerine septum to pour out of the "healthy" nostril and further confuse the picture.

The patient is asked to sit upright, and enquiry is made as to which side started to bleed first. With a head

mirror, nasal speculum and suction, the affected side is cleaned and inspected. The suction will remove clots. If bleeding is posterior, the suction tip is advanced into the nose and when the bleeder is approximated it will suddenly fill up with blood. The suction tip will thus act as a rough gauge to the depth of the bleeder. If no suction is available, the patient is asked to blow his nose to dislodge the clots. Inspection will now usually disclose anterior septal bleeders and, with luck, bleeders farther back may also be located. A small cotton fluff soaked in 5% cocaine is inserted with bayonet forceps to anaesthetise the area and reduce bleeding. The bleeding site may be verified by rubbing the area with a cotton swab. More thorough inspection is now possible.

If the bleeder is anterior and septal (Little's area), silver nitrate cautery is the treatment of choice. The silver nitrate bead is held against the vessel for 20 seconds. Actively bleeding vessels cannot be stopped in this way and electro-cautery is suggested. This will require more thorough anaesthesia. A dab of Bacitracin or any broad-spectrum antibiotic ointment is smeared on the cauterized area and the patient is instructed not to blow his nose. Care must be taken (especially with electrocautery) not to cauterize apposed areas on the two sides of the septum, as this may lead to septal perforation[3].

Cautery is of limited use in controlling deeper bleeding. Unless the vessel can be visualized, cautery is useless. When blood issues from underneath the middle or inferior turbinate, it is recommended that the turbinate be fractured out to expose the vessel[3, 4]. This may permit use of the cautery and it will certainly enhance the effectiveness of any packing against the bleeding site.

The application of direct pressure via nasal packing is the cornerstone of effective treatment of epistaxis. Anterior packing is often necessitated by severe or poorly visualized bleeding in the anterior half of the nose. Packing may be local or complete, depending on how accurately the bleeder has been localized. Nasal tampons are constructed from 3x3 inch gauze squares which are folded into strips and transfixed with long strands of black silk. These markers project from the nose and are taped to the cheek while the packing is in place. They facilitate complete removal of tampons. The packing may be soaked with an antibiotic ointment to prevent the development of a malodorous infection that usually arises in the packed nostril[3]. Sinusitis must also be kept in mind since the drainage of the sinuses is often occluded with a well-placed pack. Broad-spectrum antibiotics may be given prophylactically. The areas under the turbinates are packed first. With these special areas occluded, the nasal cavity is filled with layers of packing, built up from the floor of the cavity. Care must be taken that the packing exerts proper pressure on the walls. If tension is insufficient, bleeding persists. If the packing is too tight, pressure necrosis may ensue. This possibility is especially serious with bilateral packing of the nose[3]. Another type of anterior nasal pack is the Stevens intranasal balloon[5], which is shaped to the contours of the nasal cavity. Although it is simple to use, the Stevens balloon does not permit selective pressure at specific areas, which is possible with individual tampon packing. The balloon may be connected to a hypothermia machine, and severe epistaxis may be thus controlled. The side effect of freezing is superficial mucosal necrosis, but regeneration is rapid according to proponents of this method[2, 3].

If bleeding is frankly posterior, i.e. around the posterior edges of the inferior or middle turbinates, a posterior pack should be inserted. The stream of blood in posterior bleeding usually washes away topical agents and attempts at anaesthesia are frustrated, but cocaine flakes may provide some comfort locally. The anterior pack exerts its pressure circumferentially. When a posterior pack is properly placed, it provides a buttress against the posterior choana and allows more extensive packing of the nasal cavity. The packing can thus exert pressure not only circumferentially but also in the antero-posterior direction. Furthermore, buttressing by the posterior pack prevents the anterior packing from slipping into the pharynx which leads to gagging and eventual expulsion.

The traditional posterior pack is constructed of a small bolster of gauze which has three long ties firmly attached to it. A catheter is passed through the nose into the pharynx, its tip is grasped through the mouth with tonsil forceps and two ties from the pack are threaded through the tip. The catheter is next withdrawn from the nose, pulling the post nasal pack to the back of the throat and up into the nasopharynx. The pack is guided carefully into place, behind the free edge of the soft palate. One tie is left to protrude out through the mouth, and this is used to eventually remove the pack. The pack is firmly seated against the choana and the two ties are pulled out through the nose with the catheter. An assistant now exerts tension on the strands while the nose is packed anteriorly. The ties are fixed by knotting them around an anterior bolster outside the nostril. This exerts countertraction, holding the posterior pack in place.

The procedure sounds uncomfortable, and it is. There are several dangers inherent in posterior packing of this type. If the pack is too long, it may descend into the pharynx and stimulate gag. If the pack is too wide, it may occlude the ipsilateral Eustachian orifice and give rise to middle ear symptoms. Prophylactic antibiotics are routinely given to prevent this. The pack may also occlude the "innocent" nostril posteriorly, causing unnecessary discomfort. If the pack is carelessly guided to its niche, it may damage the soft palatal edge. Finally, anterior counteraction on the ties must be firm and constant yet not so severe as to produce external nasal necrosis.

In recent years, the Foley urinary catheter has been adapted to rhinological use and it is utilized as an effective posterior nasal pack[6, 7, 8]. The tip of the catheter is

cut off so that the balloon is at the extreme end. The catheter is lubricated and passed along the floor of the affected side until the tip is visible at the back of the throat. It is then inflated with 7 cc. of air, water or radiopaque medium, and gently drawn back into the choana. When resistance is felt, another 8 cc. are added to firmly wedge the balloon into the choanal orifice. Work with cadavers has shown that the balloon does not occlude the choana completely, yet it offers firm support to anterior packing[10]. A recurrent problem with the Foley catheter in this context has been the pressure necrosis produced by counteraction at the ala and limen nasi. This is especially common where the anterior part of the catheter is fixed by simply knotting the tube outside the nostril and cushioning the alar rim with gauze[9]. A simply constructed aluminum mask can dissipate counteraction forces over more amply vascularized parts of the face[10]. Anterior packing then proceeds as with the conventional postnasal pack.

An ingenious variant on the postnasal pack was devised in 1964 by Fortay[11]. With the aid of a catheter, a rubberized tape band is threaded in one nostril, around the posterior septal edge and out the other nostril, acting as a backstop to bilateral nasal packing. This method avoids Eustachian orifice occlusion and reduces discomfort.

Some especially refractory cases of epistaxis may bleed through the pack. In such cases it is suggested that the nose be repacked, since very often the original packing was improperly done. If the patient repeatedly bleeds through, the next line of intervention consists of more aggressive "invasive" methods.

Posterior bleeding usually originates from the sphenopalatine artery and one should attempt to block this vessel at the pterygopalatine fossa[12]. Three ml. of 2% lidocaine with 1:10,000 epinephrine are injected through the greater palatine foramen, just medial to the third maxillary molar. A No. 23 spinal needle is inserted into the foramen and advanced 28 mm. deep in the greater palatine canal. The effect of the injection is probably due to vasospasm from the epinephrine and also perhaps to mechanical compression of the artery by the bolus of lidocaine pressing on it. Caution must be exerted to withdraw the plunger first, lest one inject into the artery. Also, the needle should not be advanced any deeper than 28 mm, otherwise the foramen rotundum (at 35-36 mm), the inferior orbital fissure or the orbit itself (at 40 mm) may be entered. Padrnos has reported very good results in a small series of cases and, although it sounds forbidding, he emphasizes that even an intern can block the artery after minimal briefing.

Frank surgical intervention consists of ligation of the ethmoidal arteries (periorbital facial incision), ligation of the maxillary artery through the maxillary antrum (Caldwell-Luc approach) or ligation of the external carotid artery in the neck. Due to the extensive collateral circulation, both external carotids can be tied with no untoward effects. However, accidental ligation of the internal carotid can be a fatal mistake. Since the external carotid has branches in the neck while the internal carotid has none, it is suggested that the operator identify at least two branches of the artery before ligating. The details of arterial ligation are well covered in the literature[13, 14, 15].

Secondary Management

It is assumed, then, that the immediate problem of epistaxis is under control. What about subsequent management? Therapy will naturally be dictated by findings on history and physical examination.

The patient should be questioned about duration of bleeding. Even trivial nosebleeds can result in considerable blood loss if they persist over days, and such a history should alert the physician to "shocky" sypmtoms that may be present. A hemoglobin determination may not be helpful due to initial hemoconcentration, especially in severe epistaxis of short duration. Replacement of fluids or blood should be considered in the light of findings.

The patient should next be questioned about events immediately preceding the onset of bleeding. This will allow a broad classification of *spontaneous* and *induced* epistaxis to be made. Epistaxis may follow trauma of various sorts. Included here are rhinological operations, adenoidectomies in children, severe blows to the head or face and repeated nose blowing or picking. Also pertinent data are the time of year (nosebleeds are more common in winter), and the time of month in women (due to occasional "vicarious menstruation", which is epistaxis coincident with menstruation)[16].

The commonest cause of epistaxis is drying of the septum. In children one will often find dilated vessels in Little's area which may bleed repeatedly, only to be completely eliminated by silver nitrate cautery. Nasal blood vessels are poorly protected by surrounding tissues and are particularly prone to injury through drying. This is most prevalent in the winter with forced air heating. The drying effect will be accentuated with a septal deviation, where a particular septal area may be overly exposed to room air. The use of a humidifier should always be suggested and, where warranted submucous resection should be recommended. Intractable nose picking can be a problem in children or patients with a low I.Q. Suggested treatment here is loosely packing the nostrils with tetracycline gauze and taping the external nose shut for a week[17]. Frequent nose blowing can be traced to frequent colds, allergies or habit; this should be discouraged. A decongestant will often relieve the feeling of nasal fullness that induces repeated blowing.

More severe trauma in the form of fronto-nasal fractures can present with epistaxis as a late complication[20]. Blood from injured or fractured sinuses may pass into the nose and present as "epistaxis" at a later time. A history of trauma is therefore pertinent and should be elicited in detail.

Epistaxis is also a complication of systemic infec-

tions. Childhood exanthems such as varicella may present epistaxis as a sequela[3, 18]. An increased incidence of epistaxis has been reported in the pre-icteric phase of viral hepatitis[19], and pulmonary tuberculosis may also be accompanied by epistaxis[16].

It has already been mentioned that posterior epistaxis is more common in older patients. The reason for this is simple. In young patients epistaxis is due primarily to extrinsic factors and thus afflicts the more exposed anterior nose. In older patients epistaxis often parallels the degree of vasculopathy present and thus reflects an alteration in vessel wall structure. Atherosclerotic arteries in the nose are inflexible and prone to split open. The different types of arteriosclerosis operant elsewhere, such as Monckeberg's sclerosis, should be borne in mind during the work-up. A history of claudication or angina may be relevant as indicators of general vascular integrity.

Systemic estrogen has been used to treat epistaxis due to pathologically altered vessel walls. The hormone acts by influencing the sol-gel state of mucopolysaccharides in the ground substance of smaller vessel walls. Jacobson links epistaxis and menstrual bleeding through altered estrogen levels in the body[16]. He also attributes increased incidence of intramural hemorrhage in atheromatous coronary vessels (observed in males) to low circulating estrogen levels.

The next factor that can predispose to epistaxis is increased pressure in the vessels. It has been repeatedly shown that hypertensive patients tend to bleed from the nose and it is easy to understand how the older person with high blood pressure, vessel wall damage and external trauma is triply plagued. The correlation between hypertension and epistaxis has recently been questioned[22]; certainly, if hypertensive patients are questioned the number giving a positive history of epistaxis may indeed be statistically insignificant. However, patients do have hypertension with significant frequency and from our point of view this approach is the valid one.

Finally in the history we must enquire about blood dyscrasias and other evidence of the bleeding diathesis. In this context, the story of nasal bleeding is the story of bleeding anywhere in the body. Nasal bleeding may be due to thrombocytopenia or any of the clotting disorders which include chronic hepatic or renal failure. Physical examination must include a thorough search for purpura or any other manifestation of bleeding into the skin. Vitamin K is suggested as part of therapy and topical thrombin has also been used.

The workup of the patient should be organized to exclude:

1) *external* insult to the vessel;
2) *structural faults* in the vessel, including the abnormal vasculature of tumours or polyps;
3) *internal* stress on the vessel (hypertension, polycythemia, includinig the hperemia of systemic infections; and
4) faulty clotting mechanism of the blood *within* the vessel.

Using this system, the cause or predisposing factors of persistent or spontaneous epistaxis are usually found. Therapy can then be logically planned. Light sedation is routinely given to the overly distressed patient.

Two specific causes have been purposely left until now because their interest and importance might otherwise be unappreciated. The interesting case is nasal hemorrhage due to hemorrhagic hereditary telangectasia (Rendu-Osler-Weber disease) where the entire anterior septal surface may bleed severely and repeatedly. A split-thickness skin graft from the thigh can be used to cover the afflicted areas, and this procedure (septal dermoplasty) has "cured" most patients, at least of this manifestation of their disease[3, 17]. The important case is severe posterior hemorrhage due to a ruptured aneurysm of the internal carotid artery[21]. The rupture may be due to erosion or to trauma, or simply to hypertension. It can be picked up by selective arteriography and presents a grave exception to the glib statement that nosebleeds are never fatal.

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Beethoven And Deafness

David Silverberg, 7T3

In the end he heard nothing, but was hearing things that many men would trade their souls to hear. Still he said enough to impart immortality on the human race. So it strikes me as being strange that Beethoven, like you or me, had ears. But he did have ears, terrible ears for him, but for us they were good enough. As a matter of fact, I'm quite certain that he could never have expressed himself as he did had he not been deaf. Old Dr. Marage, who died before my time, seems to agree with me, saying that Beethoven had a deafness of labyrinthic origin and the nature of his deafness which cut him off from the outer world "maintained his auditory centres in a state of constant excitement, producing musical vibrations and hummings that he sometimes perceived with the utmost intensity — if it suppressed the external vibrations, it augmented the internal." So, out of his solitude and the torment of the deaf, we have inherited his music. (Dr. Marage felt that had otosclerosis been the cause, without any irritation of the labyrinth, his auditory nerves would have silently died away and he might never have achieved what he did.)

This throws us into the centre of the problem of Beethoven's deafness and indeed any man's deafness. Was it conductive (thus most likely middle ear in origin) or was it sensorineural (labyrinthine or auditory nerve)? Was Beethoven chasing some elusive but beautiful illusion that his deteriorating cochleas were accidentally sending out? There are many other ideas on the subject. It is said that Beethoven was unable to co-ordinate his body to music. He couldn't dance, he couldn't sing on key, he couldn't play a stringed instrument in tune. But he certainly is noted to have been one of the greatest pianists of all time, always playing with a certain intensity and gentleness which were very extreme and considered revolutionary to the times. In fact, Beethoven was a whole revolution in music and also the conduct of a composer. Whereas it was a necessity for an Artist to come to the feet of rich patrons, in Beethoven's time the commercial industry of printing music allowed him the luxury of being proud in his genius. Whereas Mozart and Haydn before him were "puppets to the Prince", Beethoven did not have to endure much of this, partly because of the times and partly because of his nature. As a sloppy youth, he said "I will not have to be a gentleman". As one who did not take heed of etiquette in royal households, he said, "Rules are for other people, but not for me". Likewise, if he broke a rule in writing music by producing a dissonant sound or switching unexpectedly to another key, he would answer criticism with "You can't do it, but I can. I am Beethoven". He once tried to break a chair over the head of a Prince; he once walked out into the pouring rain with the manuscript of the "Appasionata", almost getting it ruined after some drunk French soldiers asked him if he could play the violin. He used to run around the courtyard in the pouring rain. He once stampeded a herd of cattle for a joke. To his haughty pride as a youngster was added the attributes of a deaf man. He was difficult, quarrelsome, suspicious and withdrawn. To the extent that these aspects of his character, shaped by his upbringing, his deafness and other medical conditions, and his many social conflicts which to us may seem trivial, influenced the content and course of his musical thought — it is important to know about them in order to be closer to his music.

But what about the nature of his deafness, which is supposed to be the subject of this article. Let us examine some of the historical data which had been assembled by an army of biographers.

One early account stated that Beethoven became deaf after falling in a rage at a tenor who had upset him by making him rewrite some music. Another attributed it to an illness which he contracted by stripping himself in front of a drafty window after overheating himself. These have no physiological basis that I know of.

In a letter of Beethoven's dated June 1801, "Know that the noblest part of me, my hearing, has become very bad. Even then it has been getting worse and worse and whether or not a cure is possible has become question . . . My affliction causes me least trouble in playing and composing, the most in association with others". This letter dates Beethoven's awareness of his deafness at about 1798; therefore its onset must have been at about the age of 27.

In another letter of June 29, 1801, Beethoven writes "For the last 3 years my hearing has been getting weaker and weaker and the cause must have been the condition of my bowels which, as you know, was already at the time wretched, but has got worse, for I have a continual diarrhoea that has made we extraordinarily weak".

Later in 1801, "My hearing has grown steadily worse for 3 years . . . My ears whistle and buzz continually day and night. In any other profession this might be tolerable, but in mine, such a condition is truly frightful. I was often in despair. Then some medical ass recommended me cold baths, but a more judicious doctor the tepid ones of the Danulu which did wonders; my bowels improved but my hearing remained the same or in fact rather got worse. To give you some idea of my extraordinary deafness, I must tell you that in the theatre I am obliged to lean close up against the orchestra in order to understand the actors and when a little way back, I hear none of the high notes of instruments or the singers, and if I be put a little further away, I do not hear at all. Frequently, I can hear the tones of a conversation, but not the words, and as soon as one shouts, it is intolerable". This is undoubtedly one of the most informative notes we have. Tinnitus of ringing character, especially in association with recruitment, suggest cochlear pathology. Likewise the possible distortion of sound. However, one cannot be absolutely certain of recruitment here, and tinnitus is known to occur with

middle ear and auditory nerve pathology. We must take note that the fact that he could not hear the high notes of the instruments is good evidence of loss of high frequency sounds, which one could not be sure if it were loss of the high notes of singers. The fact that his hearing became diminished with distance seems to point more to a conductive deficit. Thus from this note, signs of sensorineural loss, including initial high tone loss (but again there are exceptions) seem to predominate.

Over the next 16 years, his deafness progressed, so that he was quite socially handicapped. He sought help from many doctors who were not able to offer much.

In a letter November 16, 1801, "Dr. Vering has vesicatories placed on both arms. I cannot deny that the ringing and sounding in my ears has become less than usual, especially in the left where my deafness began; but my hearing has not improved and I dare not say it has not grown worse rather than better."

By the fall of 1802, at the young age of 32, Beethoven wrote his last will and testament in Hieligenstadt, so it is valid to suppose that his deafness was getting on his nerves. Things continued at a more or less steady state for the next few years. Beethoven probably still had recruitment in 1809 when because of pain, he had to put pillows over his head during the French bombardment of Vienna. In his Pastoral symphony written 1807-08, amid the thunder, lightening, and sweeping rain of the storm movement, there appears a prolonged shrill of the piccolo which would seem to have no place in a thunderstorm unless perhaps it is Beethoven's tinnitus that we are hearing.

In 1812, one of his contemporary composers noted "it was a difficult task to make him understand as one had to shout so loudly that it could be heard 3 rooms distant." In 1814, the above person remarked on hearing Beethoven play the piano part of his "Archduke" trio, opus 97, "It was not a treat, for, in the first place the pianoforte was badly out of tune, which Beethoven minded little, for he did not hear it . . . In the forte (loud) passages, the poor deaf man pounded on the keys till the strings jangled, and in the piano (soft) he played so softly that whole groups of tones were omitted so that the music was unintelligible . . . I was deeply saddened at so hard a fate."

If one could liken his playing to his speech, it seems to suggest a sensorineual type in which the subject when speaking has quite a volatile range of loudness and softness whereas one with conductive deficit tends to ramble on in a monotone. On the same level of thinking one might say that Beethoven showed no evidence of hearing better in a noisy room, a characteristic of conductive deafness. It seems that the reason for this phenomenon is the fact that people automatically "alle up" in a noisy room, and whereas the deaf listener is not all that sensitive to the distant background noise, he does gain from the increased loudness of the person who is near, talking to him.

In 1814, Beethoven was using ear trumpets made for him by Malzel who invented the metronome, which Beethoven mimicked in the second movement of his 8th. Beethoven stated, "they were not of the service to me that I expected". It's doubtful that these trumpets could render much help to any deaf man, especially to one who could not discriminate words but heard only tones. This is probably due to the fact that vowels are much longer lasting than consonants. There is also evidence that he used a wooden stick attached to the piano but this probably was only useful for perception of gross verberation.

Beethoven's conversation books, in which he had people write what they had to say, date from 1817. In February 1822 Beethoven wrote "Last night I was again attacked by ear-ache from which I generally suffer at this time of year". It seems that these pains had been present since 1810. There is no history of otorrhea. In 1824 Beethoven was totally deaf, having to be turned around to face the cheering audience after the first performance of the Ninth. He must have been in a world of his own on that night.

Lastly some segments from the report given by Dr. Johann Wagner who performed the autopsy one day after death (March 27, 1827).

"The mastoid process was large and showed no groove; its cells were large and lined with a hyperaemic mucous membrane. There were traces of the same hyperaemia in the whole substance of the petrous bone which was traversed by prominent blood vessels; this was notably the case in the region of the cochlea, the membranes spiral tract of which was slightly reddened.

"The facial nerves were particularly thick; the acoustic nerves on the other hand, were atrophied and devoid of myelin. The accompanying acoustic arteries were dilated to the extent of being wider than a crow's quill; and they were of cartilaginous (not brittle) consistency. The left acoustic nerve — that was much thinner than right, sprang from three very slender grey threads proceeding from the fourth ventricle; the right nerve was a single root, stronger and clear white in colour. Their origins from the fourth ventricle of the brain showed cerebral matter that was much firmer and more hyperaemic than the surrounding matter".

Finally, since we have a choice to relate Beethoven's deafness to an isolated condition of the auditory apparatus or to the effects of some systemic disease, mention must be made of Beethoven's general health. "Beethoven was plagued with chronic ill-health from the time of young adulthood. Chief among his non-aural maladies was a continually recurrent severe diarrhoea usually accompanied by colic, and several times, notably in 1812, leading to prostration. During the summer of 1797 (about the onset of his awareness of his deafness) Beethoven contracted severe diarrhoea which lasted 6 years and it generally was attributed to typhus. He suf-

fered headaches. Attacks of asthma caused him concern that he might have had TB from which his mother died when he was 17. All his biographers bear witness to the evidence of smallpox over his face, lesions which he bore from early youth. These show up on his death masks. In a letter to Dr. Braunhofer, May 13, 1825, we learn of an episode of hemoptysis and several months of repeated epistaxis. In 1821, he complains of a protracted episode of jaundice. During the next 6 years his liver disease advanced. The cause of his cirrhosis, according to McCabe, was due to alcohol and poor eating habits, but according to the autopsy, it appears that it was macronodular cirrhosis rather than the micronodular picture seen in alcoholic cirrhosis. Besides, how could a drunk compose such music. He had an attack of conjunctivitis in 1823. There was also arthritis. Death was due to dropsy (liver failure) with a superimposed pneumonia.

I think that the most pertinent conclusions that one can draw from this data is that Beethoven had features characteristic of both conductive and sensorineural deafness. The onset was at about the age 27 with initial gross deterioration followed by a non-relapsing chronic progression to total deafness which must have lasted about 20 years. It is quite difficult to pin down such a story to one pathological process and indeed many men who have devoted their lives to the study of hearing have come to quite different conclusions as to the etiology. I think that this may be a case when a single etiology does not exist and that the explanation lies in the fact that Beethoven may have had almost every affliction known to man including otoclerosis, typhoid fever, syphillis, and perhaps even a collagen vascular disease, and it was these processes that ganged up on him to destroy the one sense from which he derived the most pleasure — just as an expression of Fate that says, "If you are to be great, then you must suffer". He suffered greatly, and at times wanted the relief of death, yet had the love for Music and for Humanity that drove him on to write beautiful piece after piece each saying more than the one before, finally reaching the level of the late String Quartets which have never been approached by any other composer. The tragedy of such a man will always be that he didn't live another year.

As stated by Cawthorne in his famous article, evaluating the effects of deafness on Jonathan Swift, Goya, and Beethoven, "the most likely cause of a progressive deafness starting in early adult life is otosclerosis". Otosclerosis basically is boney overgrowth in the middle ear, which may distort the ossicles, produce stapedial fixation and even impinge upon the cochlea. Characteristically it produces a conductive deafness, often bilateral. Initial loss of high tones is not characteristic, however, as shown in an article by Sando and Hemenway[5], who examined histologically the temporal bones of a woman with otosclerosis who died of leukemia, that cochlear invasion by the otosclerotic process had produced atrophy of the spiral ligament, collagenous deposition, atrophy of the stria vascularis, and a decrease of hair cells and spiral ganglion cells, showing that variations in the otosclerotic process could produce different effects. But no otosclerotic process was described in the autopsy.

McCabe[3], thought that the absence of middle ear pathology in the autopsy report, ruling out otosclerosis and the fact that Beethoven was able to conduct loud orchestras and play low piano at 80 - 90 decibels without intolerable pain, thus going against cochlear pathology, meant that the cause was eighth nerve deafness which may have been due to vascular causes or granulomatous inflammation of the adjacent meninges, both of which are suggested in the autopsy. Syphillis can produce isolated deafness due to eighth nerve arteritis and a low grade meningitis. But at age 27, this is quite impossible, especially without other objective evidence of tertiary lues over the next 30 years. Beethoven did have some signs of insanity, or at least bizarre behaviour, but it should be noted that he was a miserable, deaf man with problems concerning his nephew Carl, and a childhood history of being raised by a drunk father. The autopsy report offers no characteristic features of tertiary lues and it seems that there is doubt as to whether or not Beethoven had syphillis at all. Certainly it could have happened to anybody during those times but the only evidence that Beethoven had it are some vague inferences by the biographer Thayer, some hearsay by some doctors, and the record of two prescriptions which had mercury as a constituent (mercury was not a standard treatment for syphillis till 1850) as well as some book ordered by Beethoven on venereal disease toward the end of his life.

Toxic neuritis or labyrinthitis, secondary to typhoid fever, have been put forward by many including Beethoven himself. Serous effusion in the middle ear occurs in about 5% of adults with typhoid and in more than half of children. The effusion is often not associated with perforations and may lead to later adhesive otitis. The typhoid bacillus has been cultured from this middle ear effusion. (More often the effusion is the result of eustachian tube blockage secondary to typhoid infection within the pharynx. Often, of course, there is tympanic membrane perforation and signs of acute and chronic otitis media.) It can also produce chronic liver disease and may explain much of the symptomatology and pathology of Beethoven's condition. The deafness, however, should be more acute and less protracted than Beethoven's was.

One must not ignore the possibility of an immunopathic disease of which I include ulcerative colitis, SLE, and polyarteritis nodosa which may explain the chronicity of Beethoven's gastrointestinal disease, the redness of his face, the arthritis, the conjunctivitis, the arterial and visceral pathology and the frequent abscesses to which he was subjected.

In summary, I leave the question as to the etiology of Beethoven's deafness unanswered but have analysed

(Continued on page 15)

Emergency Room Dentistry

John McDowell, D.D.S., 7T4

One need not spend much time in the average hospital emergency room before some patient presents himself with a complaint pertaining to the oral cavity. Understandably physicians frequently show little interest in these problems, but nevertheless the patient requires assistance. Very little dentistry is taught in the undergraduate medical curriculum. I can recall one histology Kodachrome slide being presented to our class, upside down. (It wasn't Harry's fault.)

Patients readily seek out the opinion of their trusted physicians regarding many matters. "My dentist wants to cut my gums off — should I let him?" is only one question commonly asked. Physicians are often consulted by dentists who wish to inquire if a certain patient is "medically fit" to withstand some specific procedure. A complete course in dentistry is beyond the scope of this presentation. However an introduction to some of the most common presenting oral complaints and their treatment is probably worthwhile to anyone in attendance at a hospital emergency room. The following classification is incomplete and based only on my impression of what is most likely to bring a patient to the emergency room.

A. PAIN ASSOCIATED WITH INFECTION

The vast majority of patients who feel that they need emergency attention are in pain. It is well to remember that very frequently pain can radiate from the mandibular teeth to the ipsilateral ear and from the maxillary teeth to the paranasal sinuses. Actually it can radiate to any part of the head and neck. Conversely pain of angina pectoris not infrequently radiates to the mandibular area. Naturally the cause of the pain should be eliminated as a primary measure with the use of analgesics playing a supportive role. Almost any pain of dental origin can be controlled with dextro-propoxyphene, 65 mg with A.P.C. (Davron compound 65®), codeine 30 mg with A.P.C. (292®) or their equivalent. Most bacterial infections readily respond to a course of oral penicillin with erythromycin a good second choice. It must not be overlooked to provide prophylactic antibiotic coverage to anyone who has had rheumatic fever and about to receive oral treatment.

(i) *Periapical Abscess*

The periapical abscess is an acute or chronic suppurative process of the dental periapical region (end of the root). It occurs as a result of infection following carious involvement of the dental pulp most often but can arise after trauma to the tooth. Pain is intense but systemic manifestations of infection are minimal and usually limited to fever and regional lymphadenitis. The acute periapical abscess is so rapidly progressive that the only radiographic evidence is a thickening of the periodontal membrane. The chronic periapical abscess with a periapical granuloma demonstrates a radiolucent area at the apex of the tooth on the radiograph. Drainage must be established by either extracting the tooth or opening the pulp chamber. The latter procedure enables one to save the tooth but endodontic (root canal) treatment by a dentist will be necessary later. Complications that follow failure to treat the periapical abscess are fistula tract formation opening on the mucosa or skin, cyst formation, osteomyelitis and cellulitis. To recognize such a troublesome culprit, one can usually follow the pain to a very carious, heavily restored or traumatized tooth, which is sensitive to percussion, heat or electric current (pulp testing instrument). Radiographic and sinus tract evidence may be in attendance. If drainage is impossible, a course of penicillin plus analgesics will be helpful.

(ii) *Periodontal Abscess*

The periodontal abscess (Fig. 1) is related directly to a deep pre-existing periodontal pocket (loss of bone down one portion of the tooth root). The gingival tissues close over the entrance to the bony pocket thereby trapping bacteria which multiply and form an abscess with the destruction of additional bone. Systemic involvement is minimal and the involved tooth is tender to percussion (when in the acute stage). Treatment is drainage, best performed by an incision of the gingiva along the long axis of the involved tooth or by insertion of a probe down into the pocket. Further treatment by a dentist to prevent reoccurences is indicated.

FIGURE 1. Multiple periodontal abscesses.

(iii) *Pericoronitis (Infected, Partially Impacted Third Molars)*

The pericoronal infection occurs around partially impacted third molars and leads to trismus and cellulitis in many cases. The partially erupted tooth develops a communication with the oral cavity, bacteria enter the pocket, multiply and produce an abscess. Treatment involves extraction of the tooth when it is orientated in such a position that it can never erupt (the majority of cases) or will never serve any useful purpose. Otherwise drainage must be accomplished by excising the soft tissues overlying the crown of the tooth.

(iv) *Cellulitis*

Cellulitis is a diffuse inflammation of soft tissues which is not circumscribed or confined to one area as

it spreads through tissue spaces and along fascial planes. Cellulitis occurs as the result of bacteria that produce hyaluronidase and fibrinolysin (commonly streptococci). Cellulitis of the face and neck usually results from some type of dental infection. The patient will feel moderately ill with fever and leukocytosis. There is a painful swelling of the involved soft tissues which are firm and brawny. Skin may be of normal colour or appear inflamed and a regional lymphadenitis is usually present. Infections arising in the maxilla cause initial swelling of the upper half of the face followed by entire facial involvement. Infections arising in the mandible produce early swelling of the lower half of the face which spreads not only upward but downward to distort the neck. Treatment is removal of the cause of infection (usually one or more teeth) plus antibiotic therapy. If left untreated, the condition tends to be self limiting as far as its spreading to involve additional tissues, with facial abscess formation and drainage through the skin. When severe cellulitis involves the floor of the mouth and neck it is called Ludwig's Angina. This type is very serious as it can spread to involve the glottis carrying risk of suffocation, invade the carotid sheath, cause cavernous sinus thrombosis and meningitis.

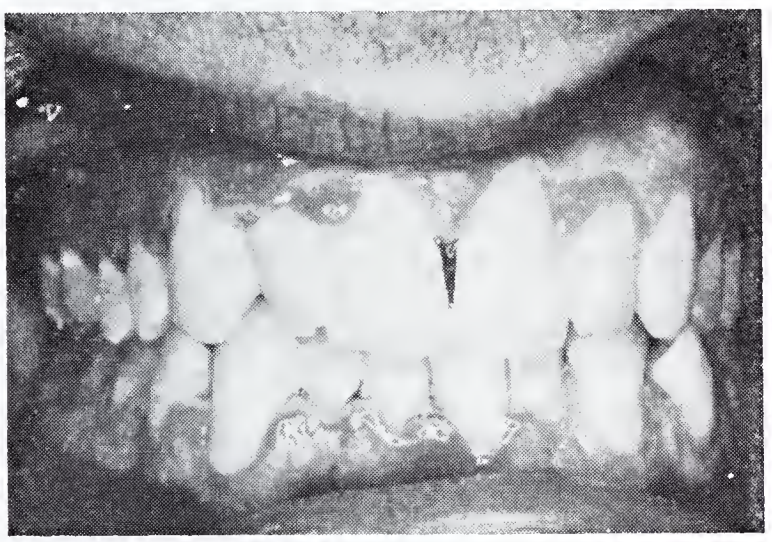

FIGURE 2. Acute necrotizing gingivitis.

(v) *Acute Necrotizing Gingivitis (Vincent's Infection, Trench Mouth)*

Trench mouth is a common specific type of gingivitis. This inflammatory process involves primarily the free gingival margin, the crest of the gingiva and the interdental papillae (Fig. 2). On rare occasions, the lesions spread to involve the soft palate and tonsillar areas (Vincent's angina). Acute necrotizing gingivitis spreads rapidly among people living in close contact but is not believed to be a contagious infection. Similar predisposing factors within the group are responsible for its occurrence, usually in young adults. Clinical features are a painful, hyperaemic gingiva with craters replacing the interdental papillae. The ulcerated remnants bleed readily and are covered with grey sloughing tissue. The picture is usually quite generalized by the time the patient seeks treatment although it begins at a single site. There is a typical fetid odour emitted and the patient will complain of pain, inability to eat, bleeding and malaise. There is usually a low grade fever and regional lymphadenitis. A fusiform bacillus and *Borrelia vincentii,* a spirochete, are always found in great numbers in trench mouth. However these bacteria as well as others found in the condition are also present in many healthy mouths suggesting that some predisposing factors are involved. Initial treatment consists of hydrogen peroxide rinses and penicillin. After 48 hours, the condition will begin to subside with adequate therapy. Thorough scaling and polishing of the teeth (with possible gingivoplasty) will be needed when the acute stage has passed.

(vi) *Acute Herpetic Gingivostomatitis*

Herpes simplex is responsible for this common condition of children and young adults. Clinical features are fever, irritability, headache, pain upon swallowing and regional lymphadenopathy. Within a few days, the mouth becomes painful and the gingiva intensely inflamed. Shortly, yellow fluid-filled vesicles develop which then rupture and form shallow, ragged, extremely painful ulcers. These are covered by a grey membrane and surrounded by an erythematous margin. The ulcers range from very tiny up to one centimeter in diameter and heal spontaneously in 7-14 days. Treatment is only supportive at the present time.

(vii) *Thrush (Candidiasis)*

Thrush is caused by infection with *Candida albicans* which is part of the normal flora of the healthy mouth. Thrush most commonly occurs in young infants, in debilitated persons, and those receiving broad spectrum antibiotics. It seems that there is a competition for nutrients between the bacteria and *Candida* which normally keeps the fungus in check. The oral lesions appear as slightly elevated soft, white plaques most frequently occurring on the buccal mucosa and tongue but can occur anywhere. These plaques resemble milk curds and are tangled masses of fungal hyphae. It is important diagnostically that these white plaques may be readily peeled off leaving a raw bleeding surface. Most other white plaque-like lesions of the oral cavity cannot be readily peeled off. Treatment is the topical administration of nystatin suspension which must be held in contact with the lesions and withdrawal of the broad spectrum antibiotic.

(viii) *Dry Socket*

The dry socket is the most common post-extraction complication and is basically a focal osteomyelitis. It occurs almost exclusively in the mandible where the bone is more dense. The blood clot disintegrates or washes out from the tooth socket leaving the bony walls exposed and there is severe pain associated with a foul odour but no suppuration. Usually onset of the pain is 24-48 hours after the tooth extraction but may not occur until a week post-extraction. Diagnosis is best made from the typical history. Treatment involves pack-

ing the socket with a material containing an obtundant. (½ inch iodoform gauze soaked in eugenol is good.) This dressing must be changed daily until symptoms have been relieved. Antibiotics are not necessary.

B. TRAUMA

(i) *Lacerated oral mucosa*

Lacerations of the oral mucosa are most common in children who have fallen and forced their anterior teeth to penetrate the mucosa. Diagnosis is obvious and treatment consists of debridement and suturing of the wound. Tetanus immunization must be assured.

(ii) *Fractured Teeth*

Lacerations are often accompanied by fractured anterior teeth. If the tooth root is fractured the tooth usually require extraction. This is best determined by viewing a periapical radiograph but may be otherwise apparent. Fractures of tooth crowns are best left to the dentist who will place protective cements over nearly exposed pulps, perform root canal therapy, construct temporary crowns, etc. However if there are any sharp edges protruding, they should be removed with a dental or bone burr.

(iii) *The Completely Avulsed Tooth*

Some accidents are capable of knocking out whole teeth with very little damage to their former sites. If properly handled these teeth can be reimplanted and function normally for years. It is a particularly worthwhile service to the public school age child as replacement bridgework is not generally successful until after the age of 16. The avulsed tooth should be thoroughly cleansed in sterile saline, a hole drilled through the lingual surface along the long axis of the root, the contents of the pulp canal and chamber removed, a root canal filling placed and the tooth fixed back into its original site. True, only a dentist would be expected to perform this operation. However it is important that emergency room staff be aware of the proper treatment and not let the potentially good tooth be discarded. Also the chance for success of this operation deteriorates as the time passes.

C. POST SURGICAL HEMORRHAGE

Hemorrhage is mainly preventable and follows extraction or periodontal surgery (gingivectomy and alveoloplasty). Sutures of 3-0 silk, oxidized cellulose (Gelfoam®) placed in haemorrhaging sockets and biting pressure applied to gauze sponges (45 minutes) will control most post-extraction haemorrhage. Electrocautery may be useful as is bismuth subgallate applied to the gauze sponges. It is important that the patient not rinse his mouth for the first few hours to prevent displacement of the blood clots. Post gingivectomy haemorrhage is controlled with sutures, electrocautery, special periodontal dressing (Coe Pak®) applied around the necks of the teeth and pressure gauzes. If these methods fail, the blood clotting factors may be abnormal due to anticoagulant therapy, liver disease, genetic factors, etc.

D. LOCAL ANAESTHESIA TECHNIQUES

For any operative procedure, 2% lidocaine hydrochloride with epinephrine 1/100,000 is suitable (the usual contraindications to these drugs apply). The epinephrine prolongs the local anaesthetic activity, reduces haemorrhage as well as the chance of toxicity from the local anaestheitc. An infiltration technique is adequate for the maxillary teeth; about 1 ml. of the solution is deposited adjacent to the apex of the particular tooth by penetration of the mucobuccal fold. The palatal mucosa is anaesthetized by infiltrating the solution opposite the apex of the particular tooth. Care must be taken as this injection is painful. A 25 gauge, 1⅝ inch needle can be used for all dental injections. Effective anaesthesia of adult mandibular teeth requires a nerve block of the inferior alveolar nerve in the following fashion. The left index finger palpates the external oblique ridge and the anterior border of the ramus of the mandible. The greatest depth of the anterior border (the coronoid notch) where the external oblique ridge joins the internal oblique ridge is at the same vertical height as the mandibular sulcus, through which the nerve enters the mandible. Thus a finger in the coronoid notch indicates the vertical level of the injection. The needle is inserted from the opposite side of the mouth so that the barrel of the syringe lies over the opposite bicuspid teeth. The mucosa of the pterygomandibular depression, just lateral to the pterygomandibular raphé is punctured and the needle gently advanced until bone is encountered at a depth of about 1 inch. The needle is slightly withdrawn and 1.5 ml of solution is deposited near the inferior alveolar nerve trunk. Then the needle is withdrawn ½ inch and a further 0.5 ml of solution is deposited on the nerve trunk of the lingual nerve. A supplemental injection into the mucobuccal fold opposite the third molar tooth anaesthetizes the long buccal nerve.

E. MINOR ORAL SURGERY

There are vast numbers of different forceps available but most teeth can be easily removed with one of the following forceps: cowhorn, hawksbill or lower anterior forceps. The latter 3 are used for mandibular teeth and have tongs that are at right angles to the shaft. The 76S is used for maxillary teeth and is a relatively straight instrument. The forceps are seated as far along the root (if exposed) as possible to minimize the risk of fracture and the tooth is luxated slowly from side to side. This motion is combined with a force directed straight out of the tooth socket and a squeezing of the forcep handles (particularly when using a cowhorn). If one is unfortunate enough to fracture the tooth, a straight elevator works well when used as a lever against the adjacent tooth or its bony support. If this fails it may become necessary to raise a flap of muco-periosteum in order to remove some of the alveolar bone. An incision is made following the necks of the adjacent teeth on either side and the proximal end extended laterally towards the depth of the mucobuccal fold. This flap of mucoperios-

teum is reflected and retracted utilizing a periosteal elevator, bone is removed with a bone burr or mallet and chisel and the root tip elevated out. The socket is curretted, rough bony edges filed and silk sutures placed. The secret to working in a mouth as elsewhere is a good visual field accomplished by adequate lighting, retraction and suction. Biting pressure on a gauze sponge for 45 minutes should control haemorrhage. The mental foramen lies between the apices of the first and second mandibular bicuspid teeth and is best left undisturbed. The maxillary sinus is separated from the maxillary posterior teeth by a paper thin layer of bone. Care must be exercised so that root tips are not pushed up into this sinus. They are very difficult to retrieve and may precipitate maxillary sinus complications.

If a tooth presents with a periapical abscess and one wishes to preserve the tooth, drainage should be attempted through a coronal hole into the pulp chamber. If this fails to achieve drainage, a vertical incision is made through the mucobuccal fold at the level of the apex. A window may have to be opened in the buccal bony plate if it doesn't already exist and a T drain sutured into place.

Many of the procedures outlined above may seem inappropriate or impossible as most emergency rooms are not equipped with the dental instruments described above. However the major expensive items, chair, lighting, suction, etc., are provided in E.N.T. areas and the missing instruments are few and inexpensive. Minor emergency dental treatment can be easily perfected with a little experience and the recipient patients are most grateful indeed. Of course it is ideal if a dentist is available to perform the best possible treatment but in an emergency the medical staff may be of valuable service. Naturally it is impossible to cover much oral pathology in this paper but one must be aware that most of the pathological processes operating elsewhere in the body are also found in the mouth.

ACKNOWLEDGEMENT

Photographs were provided through the courtesy of Dr. Ron Golden.

BEETHOVEN (cont'd. from page 11)

previous thought on the matter much in the same way as Dr. Douglas Snell at the Toronto General, to whom I would like to express my appreciation. Once again, I would like to thank LVB for writing his music.

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Anthony F. Jahn, B.Sc., 7T4

Embryological Development Of The Human Ear

The human ear is complex in function. Its embryological development is also complex, involving each of the three primary layers and extending well into the neonatal period. This discussion will deal with the three divisions of the ear and its casing, the temporal bone. The neurohistological development of the inner ear will not be considered since this itself would merit an entire article.

At the age of two weeks, when the first somites appear, the human embryo is a tiny plate of tissue lying on top of the yolk sac and in turn surrounded by the amniotic sac. At this stage the embryo shows clear differentiation of the three embryonic layers, ectoderm, mesoderm and entoderm. The adult ear takes partial origin in each of these three layers. Beginning in the third week of development there may be seen an ectodermal thickening on either side of the neural crest which is the *otic placode*. Seen from the side, the otic placode seems to sit in a niche, bounded by the neural fold dorsally, the first somite posteriorly and the developing branchial arches ventrally. Its position is more or less directly in line with the second branchial cleft.

The otic placode is thus framed by structures which limit its surface expansion. In the third and fourth weeks the ectoderm of the otic placode proliferates and it accommodates this increase in surface area by dimpling and invaginating. The otic pit thus formed continues to expand inward. The lumen of the structure grows more cavernous and its surface connection more tenuous, until it detaches itself from the surface and begins migrating medially as the *otic cyst* or *vesicle*.

During this time the primitive foregut is developing into the embryonic pharynx with *branchial arches, clefts* and *pouches*. These structures are also known as pharyngeal or visceral arches, clefts and pouches. It should be noted that the primitive pharynx is mesodermal in substance, with an ectodermal covering and an entodermal lining.

Four pairs of arches may be seen in the embryo, with the fifth arch buried above the epipericardial ridge and not visible on inspection. Each arch contains three structures. In the centre of each arch is a bar of local mesodermal condensation which is the *cartilage bar* of the arch. Posterior to the cartilage bars are nerves which course ventrally from the primitive neural chord. Behind the nerves are blood vessels, communicating arteries between ventral and dorsal aortas. Of particular interest are the cartilage bars of the first and second branchial arches, known as *Meckel's* and *Reichert's cartilage* respectively. Also of interest are the arteries contained in the second and third branchial arches which subsequently develop into the stapedial and internal carotid arteries, in that order. It will be recalled that the relevant vasculature of the embryo at this stage consists of a ventral aorta, paired dorsal aortas and branchial arteries connecting them.

As the primitive pharynx continues differentiating, the first pharyngeal pouch on either side expands laterally to form the *tubotympanic recess*. This occurs simultaneously with the medial migration of the otocyst mentioned previously. On the external surface, the first pharyngeal cleft, which lies directly opposite to the developing tubotympanic recess, hollows out to meet the approaching tubotympanic cavity. They remain separated by a thin septum. The gross outline of the ear can now be visualized, with the otocyst precursor to the inner ear, the tubotympanic cavity destined to house the middle ear structures and the inward hollowing of the first branchial cleft representing the primary external ear canal (Fig. 1).

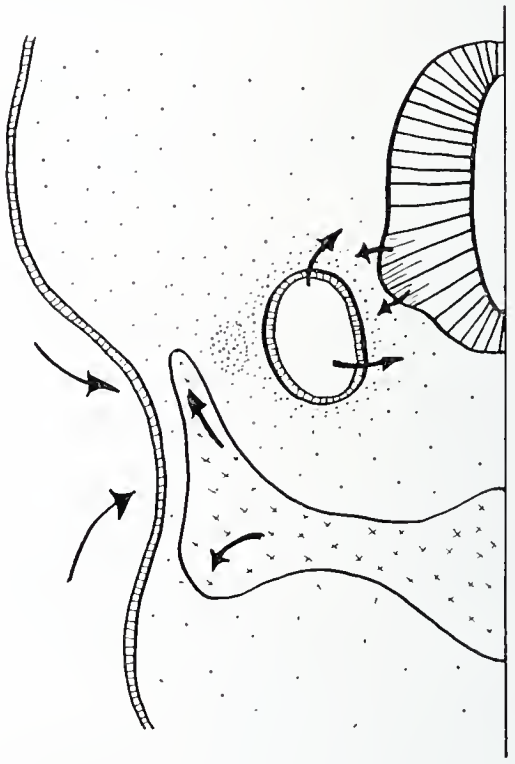

FIGURE 1
Early development of external and middle ear regions. The otocyst has taken up position dorsomedial to the expanded extremity of the tubotympanic recess.

During the medial migration of the otocyst, there arises a localized thickening on the lateral aspect of the neural tube. This represents the *acoustico-facial neural crest primordium* which will give rise to the ganglia of nerves VII and VIII. The otocyst approaches this neural thickening and becomes surrounded by a condensation of mesenchymal tissue, the precursor to the otic capsule. The lateral extremity of the tubotympanic recess continues to expand below and beyond the otocyst which is now medial to this cavity, and the future anatomical relationship of the middle and inner ear is defined. The acoustico-facial neural crest primordium detaches itself from the neural tube, approaches the otic vesicle and splits to form the future facial ganglion dosally and acoustic ganglion ventrally.

At this point, the end of the fourth week of development, let us concentrate on the otocyst. This structure is an ectodermally derived fluid filled sac. It lies close

to the neural tube, dorsomedial to the expanded end of the tubotympanic hollow. By 4½ weeks the blastema of the future stapes is also recognizable as a condensation of mesenchyme just lateral to the otocyst. At the end of four weeks, the spherical otocyst begins to assume a dorso-ventrally enlongated shape. The ovoid vesicle invaginates on the lateral side and the part of the cyst above this invagination elongates further, the dorsal extension continues, bends medially, and soon is recognized as the future *endolymphatic duct and sac*. The cyst also elongates ventrally forming the *cochlear extension* of the future membranous labyrinth. The extension continues to elongate and begins to curl to eventually assume the snail-like shape of the cochlea. A second invagination on the medial side of the organ now separates the main body into a saccular and utricular segment.

As the endolymphatic duct and cochlear extension continue their development, a third invagination may be seen on the lateral side of the labyrinth. Along with the second invagination this forms a ring-like construction around the middle of the developing inner ear. As well, the dorsal segment of the structure develops saucer-like extensions in three planes. These extensions grow biconcave and central resorption begins which will eventually result in the formation of the definitive semicircular canals (Fig. 2).

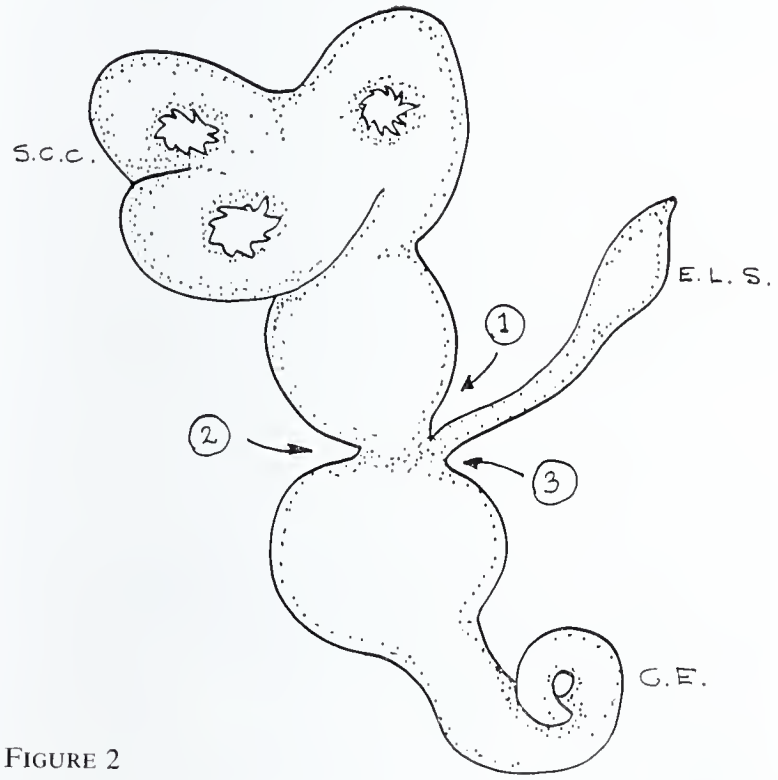

FIGURE 2

Developing membranous labyrinth, with endolymphatic duct and sac (E.L.S.), cochlear extension (C.E.) and semicircular canals (S.C.C.). Numbers indicate chronological sequence of invaginations (see text).

The final stage of gross development of the inner ear occurs as the cochlea completes 2½ turns and the annular constriction about the middle of the organ grows tighter to form the *ductus reuniens* joining the cochlear labyrinth to the rest of the membranous inner ear. The vestibular, cochlear and endolymphatic duct parts of the membranous labyrinth are now joined by a 'Y' shaped duct. The acoustic and vestibular nerves approach and join the membranous labyrinth.

Let us now consider the formation of the auricle. In the fourth week the first pharyngeal pouch invaginates to form the *primary external auditory canal*. This approximates the lateral extension of the tubotympanic recess which extends laterally from the pharyngeal lumen. The external ear appendage itself develops from the first pharyngeal cleft and adjoining aspects of the first and second pharyngeal arches. It should be briefly mentioned that the second pharyngeal arch grows posteriorly in the fourth week to come in contact with the epipericardial ridge. In this way the third and fourth arches come to lie deep to the surface, and a sinus is formed. This is the *cervical sinus of His*, of clinical importance because it may persist and drain in any of several directions, to the surface or into the pharynx.

In the sixth week the apposed sides of first (mandibular) and second (hyoid) arches develop sub-surface thickenings called *hillocks*. There are three hillocks seen on each arch and they are numbered clock-wise from No. 1, the ventral mandibular hillock to No. 6, the ventral hyoid hillock. There is next seen a generalized proliferation of the hyoid arch elements forwards with a resultant fusion of the hillocks to form the external ear appendage. By 12 weeks the hillocks are completely fused except for the ventral mandibular hillock (No. 1), which remains separate to give rise to the tragus of the adult ear (Fig. 3).

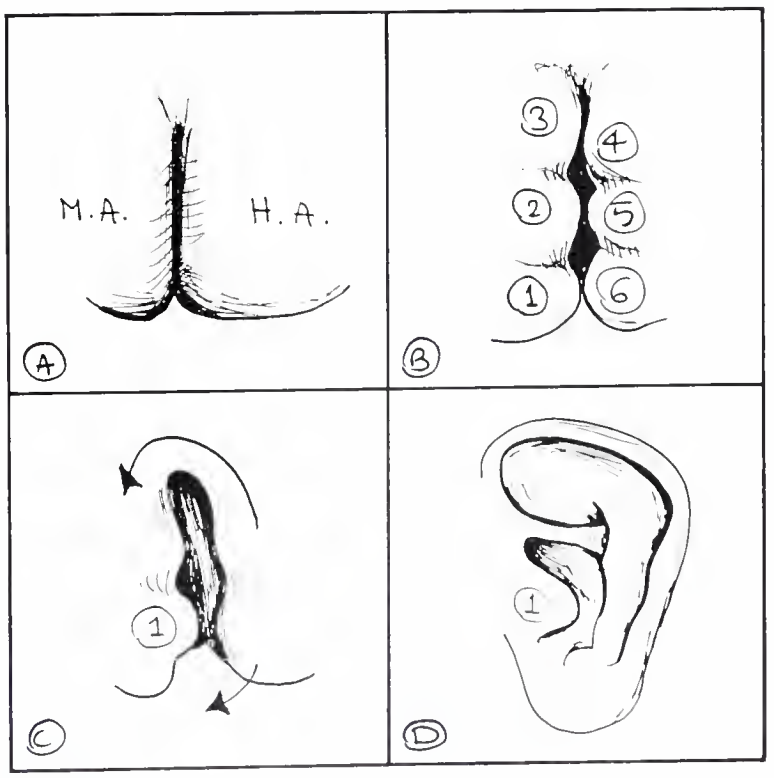

FIGURE 3

Development of the auricle from adjacent aspects of mandibular and hyoid arches. M.A. — Mandibular arch; H.A. — Hyoid arch.

We will now consider the formation of middle ear elements. As previously mentioned, in the 4½-week embryo the blastema of the stapes is already recognizable as an area of mesenchymal condensation. If we examine the cartilage elements of the pharyngeal wall (Fig. 4A), we will see that this blastema is merely the dorsal aspect of Reichert's cartilage (2nd arch) that has prolif-

erated and grown posteriorly. One should also note the disposition of Meckel's and Reichert's cartilages and *the interbranchial cartilage bridge* that connects them. Recall also the relations of the nerve and artery of the second arch which are the stapedial artery and facial nerve. As the facial nerve courses lateral to the "head" of the cartilage bar, it divides the cartilage into three parts. The *laterohyale* blastema lies anterior to the nerve, and *interhyale* lies medial to the nerve and the *stapes blastema* (apposed to the stapedial aftery) lies posteriorly. The chorda tympani passes ventrally and deep to the interbranchial bridge suggesting its eventual pathway around the neck of the malleus.

It is classically taught that the ossicles are derived from three sources: Meckel's cartilage (of the first arch), Reichert's cartilage (of the second arch) and the otic capsule. The cartilagenous anlage of the ossicles is actually formed from the interbranchial bridge (malleus and incus) and caudal extension of Reichert's cartilage (stapes). The inner layer of the stapes footplate arises from the otic capsule and the anterior process of the malleus is the product of independent intramembranous ossification. The cartilage models of the ossicles can first be visualized as the interbranchial bridge develops a longitudinal groove, to define the *malleus blastema* ventrally and the *incus blastema* dorsally. If the first branchial cleft is projected on this cartilage bridge to divide it into first and second cartilage elements, it will be seen that both of these ossicles are derived from both cartilages (Fig. 4A).

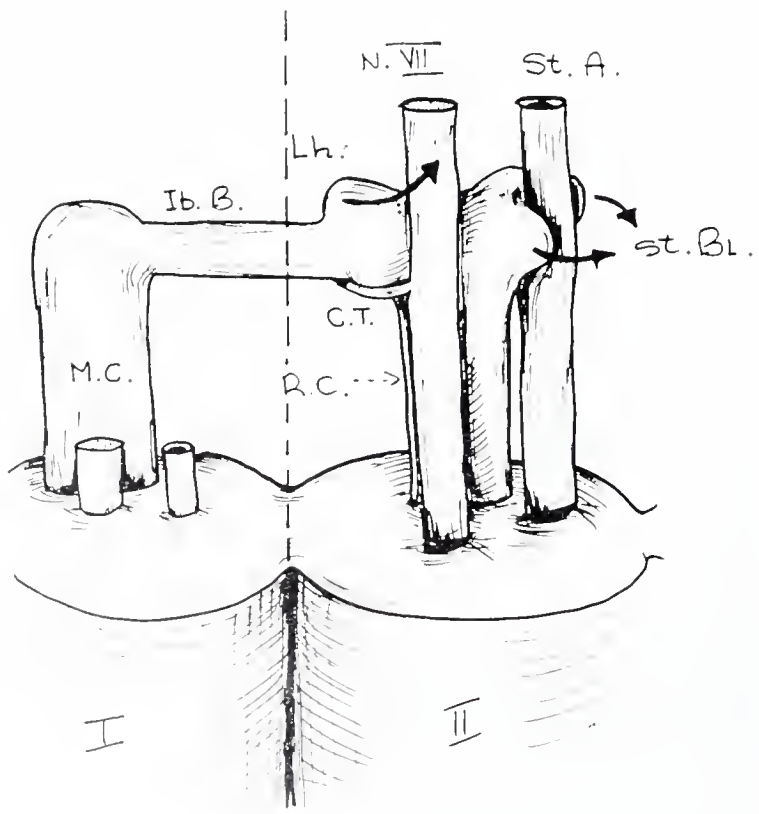

FIGURE 4A
Relationship of Reichert's cartilage to stapedial artery, facial nerve and Meckel's cartilage. M.C.: Meckel's cartilage, Ib.B: Interbranchial bridge, Lh: Laterohyale blastema, R.C.: Reichert's cartilage, N.VII: facial nerve, C.T.: chorda tympani, St.A.: Stapedial Artery, St.B.: Stapes blastema. Dotted line indicates division of interbranchial bridge by first branchial cleft.

During this time, the laterohyale and stapes blastema proliferate around the seventh nerve, to accentuate the notching of the cartilage by the nerve. The stapes blastema also grows backwards to surround the stapedial artery. This tendency continues as the laterohyale surrounds the facial nerve to form part of the future *facial (Fallopian) canal*, and the stapes blastema forms a complete ring around the stapedial artery. At eight weeks of development the stapedial artery is surrounded, and the postero-medial aspect of the stapes ring *(annulus stapedius)* flattens to form the outer part of the future footplate (Fig. 4B).

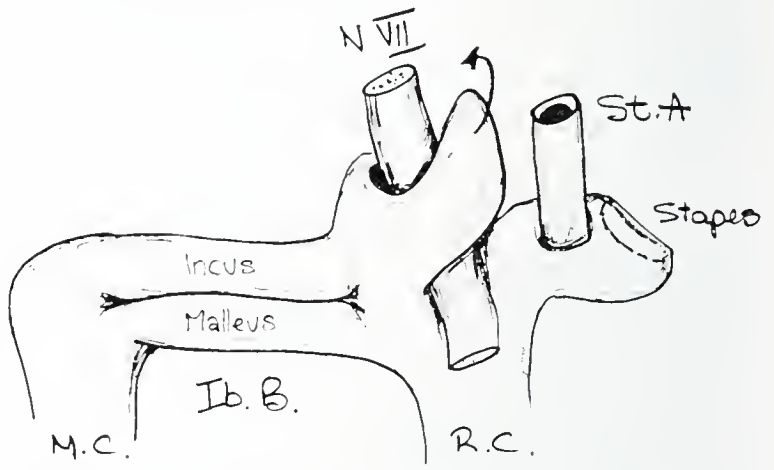

FIGURE 4B
Modification of interbranchial bridge, laterohyale blastema and stapes blastema to form anlage of ossicles and of Fallopian canal.

By the ninth week, recognition of ossicular details is possible. The head and neck of the malleus and body and short process of the incus are derived from Meckel's cartilage, while the manubrium of the malleus and the long process of the incus are formed from Reichert's cartilage. The stapes is also recognizable with stapedial artery passing between the crura and the interhyale cartilage differentiating to form the tendon of the stapedius muscle. The laterohyale cartilage is transformed in part to envelop the facial nerve and in part to form the pyramid and attachment of the stapedius muscle. The stapes footplate, defined as the flattened posterio-medial aspect of the stapes ring, approximates the oval window region of the cartilagenous otic capsule. There is fusion of the flattened aspect of the annulus stapedius and the *lamina stapedius* of the otic capsule to form the definitive footplate anlage.

The stapes footplate is anchored into the oval window of the developing bony otic capsule by means of the annular ligament. Immediately deep to the stapes footplate in a cephalic direction there develops a track of perilymphatic tissue known as the *fissula ante fenestram*. This fissula is in continuity with the vestibule deep to the footplate, the annular ligament, and the middle ear via a small dimple on the otic capsule. It may represent a site of perilymph absorption and its clinical significance lies in the fact that it may be a primary focus of otosclerosis. A similar tiny extension behind the oval window is present in two-thirds of embryos and is called the *fossula post fenestram*. Its significance is unknown.

The cartilage models of the ossicles are now complete and calcification begins at 15-16 weeks for the malleus and incus and 18 weeks for the stapes. The malleus ossifies from a single centre on the medial surface of its neck. At the same time Meckel's cartilage begins to degenerate to form the anterior ligament of the malleus. The incus ossifies from a single centre located on the anterior surface of the long process. The stapes is last to ossify, and it begins from a single centre on the obturator surface of the footplate. Osteogenesis proceeds along the obturator surfaces of the crura. The stapes differs from the other two ossicles in several respects. Ossification is irregular, and the lamina stapedius derived from the otic capsule normally remains cartilagenous throughout adult life. The stapes also differs from the other ossicles (and long bones in general) in that there is no remodelling and no increase in size with body growth. The stapes actually loses bulk while attaining adult form. Occasionally the stapedial artery may persist vestigially into adult life.

Let us now return to the external ear. It will be recalled that in the late five-week embryo the first pharyngeal groove has invaginated and is separated from the lateral recess of the tubotympanic cavity by only a thin membrane. While the tympanic cavity enlarges and grows more complex, the external auditory meatus becomes occluded by a mass of ectodermal cells which are derived from the adjacent pharyngeal arches and migrate inward to block the primitive canal. At eight weeks a definite occluding plug called the *meatal plate* is seen. A cross section of the 8½ week embryo clearly demonstrates the cartilage walls of the external auditory meatus (Fig. 5). The meatal plate extends ventro-medially below the lateral reaches of the tubotympanic recess. The primitive tympanic cavity extends between the anlage of the malleus laterally and of the stapes medially.

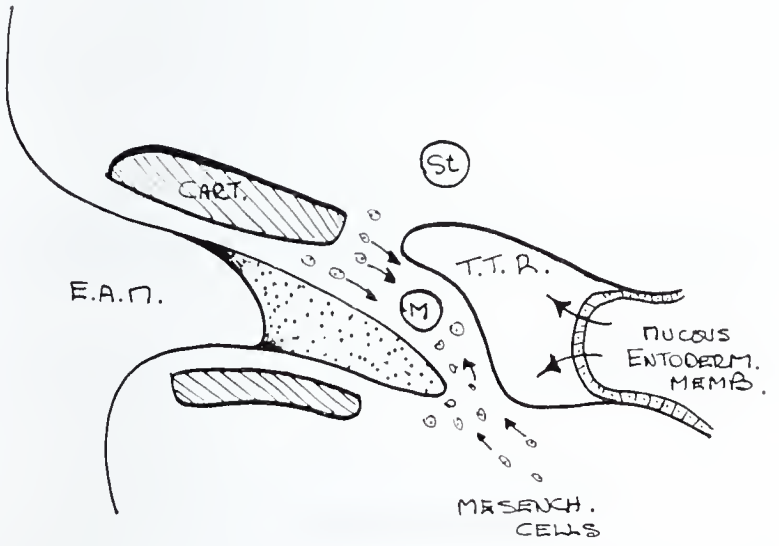

FIGURE 5
Relationship of external auditory meatus (E.A.M.) with meatal plate, developing tympanic membrane, with malleus (M) handle medially. Note external auricular cartilage (Cart.), mesenchymal invasion around malleolar handle, and invading mucous entoderm, approaching the middle ear cleft through the Eustachian tube.

The thin septum separating external and middle ears is now invaded by mesenchymal tissue. This tissue is destined to form the middle fibrous layer of the definitive tympanic membrane.

In the 21st week of development the meatal plate begins central disintegration. The peripheral cells of the meatal plate remain adherent to the wall of the tympanic septum. This ectodermal layer, along with the fibrous mesenchyme deep to it and the entodermally derived mucous layer that later coats the inner aspect of the septum, together form the tympanic membrane. The ear drum is thus seen to be derived from all three embryonic layers.

Meanwhile, the excavation of the middle ear cavity has also begun. During the early development of the middle ear, the tympanum consists of two parts: a lower half, derived from pharyngeal entoderm (the tubotympanic recess) and an upper half filled with loose mesenchyme. The chorda tympani divides the middle ear cleft into two halves. The cartilage models of the ossicles are suspended in the mesenchymal matrix of the tympanum. Towards the end of the first trimester the mesenchymal tissue of the middle ear grows loose and vacuolated. Simultaneously there is an extension of the mucous epithelium through the tubotympanic duct into the tympanum. This mucous membrane pushes the mesenchymal matrix back, and defines the contours of the middle ear (Fig. 6A).

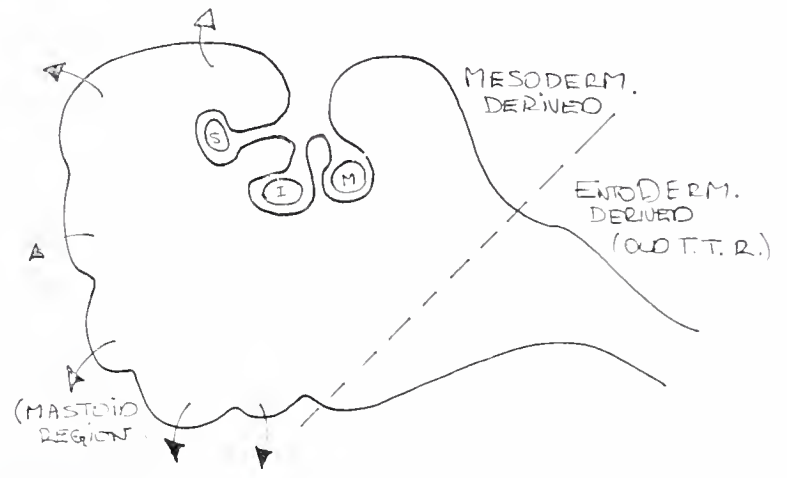

FIGURE 6A
Excavation of the middle ear cleft, mastoid antrum and mastoid process by entodermal mucous membrane.

The lining membrane envelops the ossicles which are now connected to the walls of the tympanic cavity by folds of epithelial lining. Ligaments subsequently develop in these mesentery-like folds. The epithelium plasters the handle of the malleus against the tympanic membrane to complete the formation of the ear drum (Fig. 6B).

In the 30th week of development the epitympanic space has been defined and the lumen of the antrum starts to form. In the 34th week, pneumatization of the mastoid area begins. Although the excavation of the tympanic cavity and antrum is complete by the end of the third trimester, the mastoid air cells continue their development into the neonatal period. Again, epithelial invasion is thought to play an active role in this process and this theory explains why infection of the middle ear lining may result in a poorly aerated mastoid.

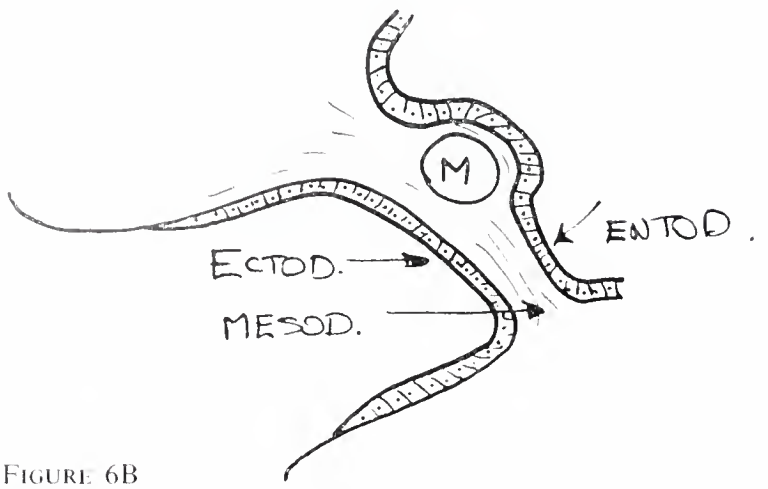

FIGURE 6B
Three embryonic layers take part in the formation of the definitive tympanic membrane.

The gross structures of the middle and inner ears are now defined in cartilage and ossification begins. The membranous labyrinth of the inner ear, as will be recalled, is surrounded by dense mesenchyme which developed as the otocyst migrated from the surface of the embryo. Between the 15th and 21st week of development this cartilagenous otic capsule begins to ossify. Ossification proceeds from 14 separate centres and by the 21st week the bony otic capsule is essentially complete.

Two areas of late ossification are noted: one is anterior to the oval window in the region of the fissula ante fenestram. Another covers a major portion of the three semicircular canals.

The ear is housed in the temporal bone, which is formed of four components: *squamous*, *tympanic*, *petrous* (petromastoid) and *styloid* bones. Each of these ossifies separately in an overlapping sequence that covers most of intrauterine life. The squamos portion of the temporal bone ossifies from a single centre which also extends into the root of the zygomatic process. This occurs in the eighth week of development. In the 10th week the tympanic ring begins ossification from a single centre, located medially and ventrally. Bone formation then ascends the two arms of the tympanic ring to form an incomplete circle of bone interrupted by a dorsal hiatus, the *notch of Rivinus* (incisura Rivini). The petrous portion of the temporal bone begins to ossify in the 24th week of development. The petrous bone ossifies at the same time as the otic capsule and like the capsule, it also ossifies from several centres.

The styloid process is ossified from two centres. At birth a centre of ossification develops at the junction of the petrous and styloid bone which fuses the two bones together. At two years of age another centre of ossification develops towards the tip of the process. The four parts of the temporal bone, in articulation with the mandible, are seen in Fig. 7.

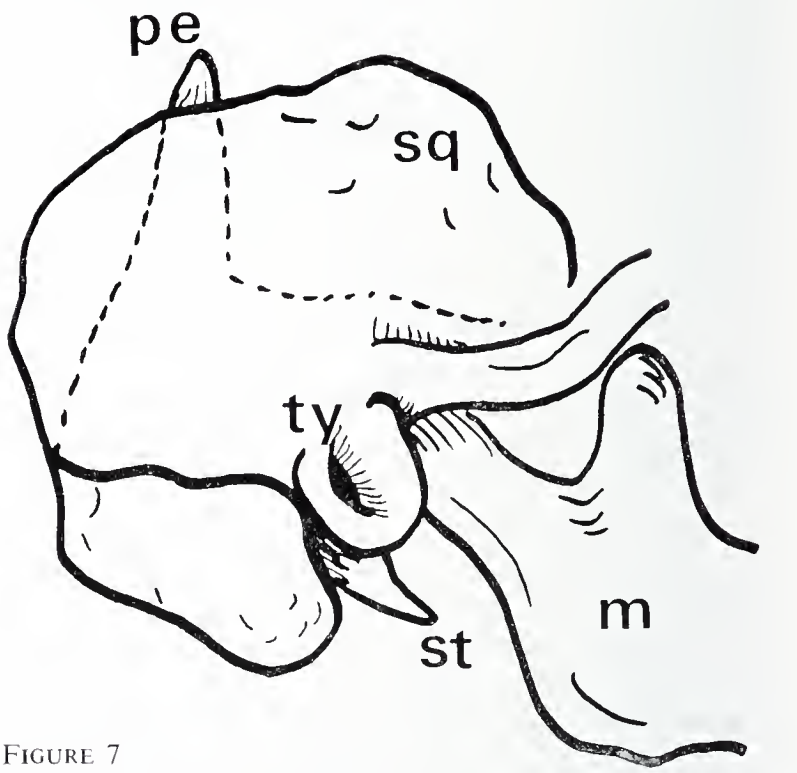

FIGURE 7
Petrous (pe), Squamous (sq), tympanic (ty) and styloid (st) portions of the temporal bone, in articulation with the mandible (m). Note notch of Rivinus at the top of the tympanic ring.

If we now consider the summary chart (Fig. 8), we will see that the development of the various parts of the ear and temporal bone occur simultaneously. It will also be noted that for each part there exist periods of intense differentiation, alternating with periods of relative dormancy. The external and inner ears are earliest to begin formation while the middle ear and otic capsule develop later. The ear does not attain adult form until some time after birth.

ACKNOWLEDGEMENTS

The author would like to thank the Temporal Bone Histopathology Laboratory of the University of Toronto for providing printed and histological background material for this article.

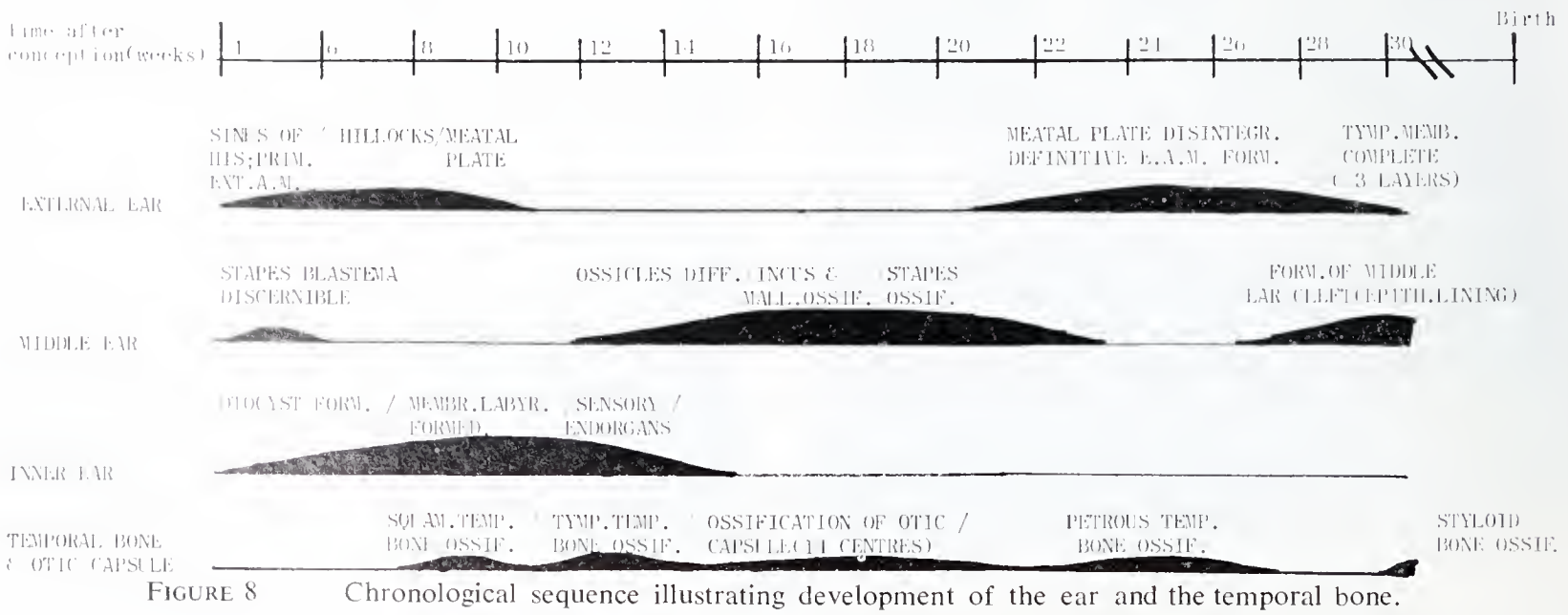

FIGURE 8 Chronological sequence illustrating development of the ear and the temporal bone.

Grand Round Quiz — Questions

Mike Ginsberg, 7T3

Select the 1 correct answer in each question. (Answers on page 22.)

1. *A distressed mother brings her 5½-year-old son to the emergency department where you are working. She tells you that the child has a severe sore throat which developed only 1½ hours ago and the nurse records the temperature at 39.5°C. You observe the child in acute respiratory distress, restless, cyanosed, with flared alae nasi and retraction of suprasternal, supraclavicular, intercostal and subcostal spaces. He is sitting, leaning forward with his mouth open drooling. On this basis, your working diagnosis is:*
   (a) acute asthmatic attack
   (b) tracheitis
   (c) acute epiglottitis
   (d) foreign body aspiration
   (e) acute diphtheric laryngitis.

2. *The appropriate initial treatment of the child in question 1 is:*
   (a) immediate tracheotomy
   (b) atmosphere of high oxygen and high humidity
   (c) ampicillin, 150 mg/kg/day
   (d) expectorant, bronchodilating agents and antihistamines
   (e) corticosteroids.

3. *A 60-year-old man born in Austria returns to your office complaining of persistent hoarse voice. He says it began 2½ months ago when he had a sore throat. At that time, you gave him a 10-day course of penicillin and advised him to quit or cut down on his 2 pack/day smoking habit. Which of the following investigations is* NOT *indicated?*
   (a) laryngoscopy
   (b) T.B. test
   (c) V.D.R.L.
   (d) x-ray examination of the larynx
   (e) A.S.O. titre

4. *Epistaxis is* LEAST *likely to be associated with:*
   (a) trauma
   (b) rheumatic fever
   (c) hypertension
   (d) leukemia
   (e) blood dyscrasia.

5. *A young mother brings her 13-month-old daughter to see you. She has a fever of 39.8°C and has been crying, very cranky and eating poorly for 2 days. She has vomited once. You find her throat clear and chest, CVS and abdominal examination normal. You find the right ear drum is reddened with loss of light reflex. Treatment includes:*
   (a) penicillin
   (b) penicillin and A.S.A.
   (c) ampicillin
   (d) ampicillin and A.S.A.
   (e) none of the above — this is a viral illness.

6. *A young man comes into your office complaining of a persistent stuffy nose, forever running. He says this has persisted for some months. Occasionally his nose itches, triggering off a bout of sneezing. On examination you find edematous turbinates and a bluish grey colour to his mucous membranes. He has no fever and functional inquiry is essentially negative. Your diagnosis is:*
   (a) common cold
   (b) deviated septum
   (c) chronic sinusitis
   (d) allergic rhinitis
   (e) none of the above.

7. *Which of the following may be associated with deviated septum?*
   (a) nasal obstruction
   (b) postnasal drip
   (c) snoring
   (d) headache
   (e) all of the above.

8. *Which of the following is not a possible complication of sinusitis?*
   (a) meningitis
   (b) osteomyelitis
   (c) cavernous sinus thrombosis
   (d) brain abscess
   (e) otitis externa.

Here are 2 questions for the basic scientist:

9. *The fast component of nystagmus is of vestibular origin. True or false?*

10. *Cold water introduced into the external auditory canal produces a nystagmus to the opposite side. True or false?*

Grand Round Quiz — Answers

1. (c) acute epiglottitis. The child's age, his sitting position, and the sudden onset and progression of his respiratory embarrassment along with the fever are very suggestive. Remember that usually the use of a tongue depressor (a laryngoscope is sometimes necessary) will reveal a large, red, edematous, cherry-like epiglottis. However, this latter manoeuvre can precipitate sudden and complete obstruction and should only be done if tracheotomy can be performed at once (i.e. ENT should be called immediately).

2. (a) tracheotomy. This child is markedly cyanosed and restless, air entry is greatly diminished bilaterally. The absence of stridor may be due to a very small tidal volume. Immediate tracheotomy is thus indicated. About half of the cases of acute epiglottitis will require tracheotomy.

   After this, an atmosphere of high oxygen and high humidity should be used, and ampicillin 150 mg/kg/day begun, since the classical etiological agent is *Hemophilus influenzae* (occasionally beta-hemolytic streptococci and pneumonococci). In acute epiglottitis expectorants, bronchodilating agents, antihistamines and corticosteroids are of no value.

3. (e) ASO titre. Laryngitis is not a complication of streptococcal pharyngitis, so the onset of hoarseness with strep throat was incidental. In this man, carcinoma of the larynx must be ruled out. TB and syphillis may occasionally present this way with little else to be found clinically.

4. (c) hypertension. There is, at present, doubt about any relation between hypertension and nosebleed.

5. (d) ampicillin and A.S.A. Although up to 50% of otitis media may indeed turn out to be viral, this child has high fever and is quite toxic, making bacterial infection more likely.

   In infancy approximately 1/3 of the cases of otitis media are due to *Hemophilus influenzae*, so ampicillin must be used. A.S.A. will help both the pain and the fever.

6. (d) allergic rhinitis. This is a classical description.

7. (e) all of the above.

8. (e) otitis externa.

9. False. The slow component of nystagmus is of vestibular origin, the fast component is believed to originate within the cerebral hemispheres. Hence, the explanation for doll's eyes reflexes in C.V.A.'s.

10. True. The easiest way to remember it is COWS — Cold Opposite, Warm Same (named by the direction the quick component takes).

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- ☐ Bioavailability in Drug Therapy, M. Pernarowski, Canadian Pharmaceutical Association Journal, February 1971.
- ☐ Drugs, Drug Products and Prescribing Habits, D.N. Wade, Drugs, No. 2, 1971.
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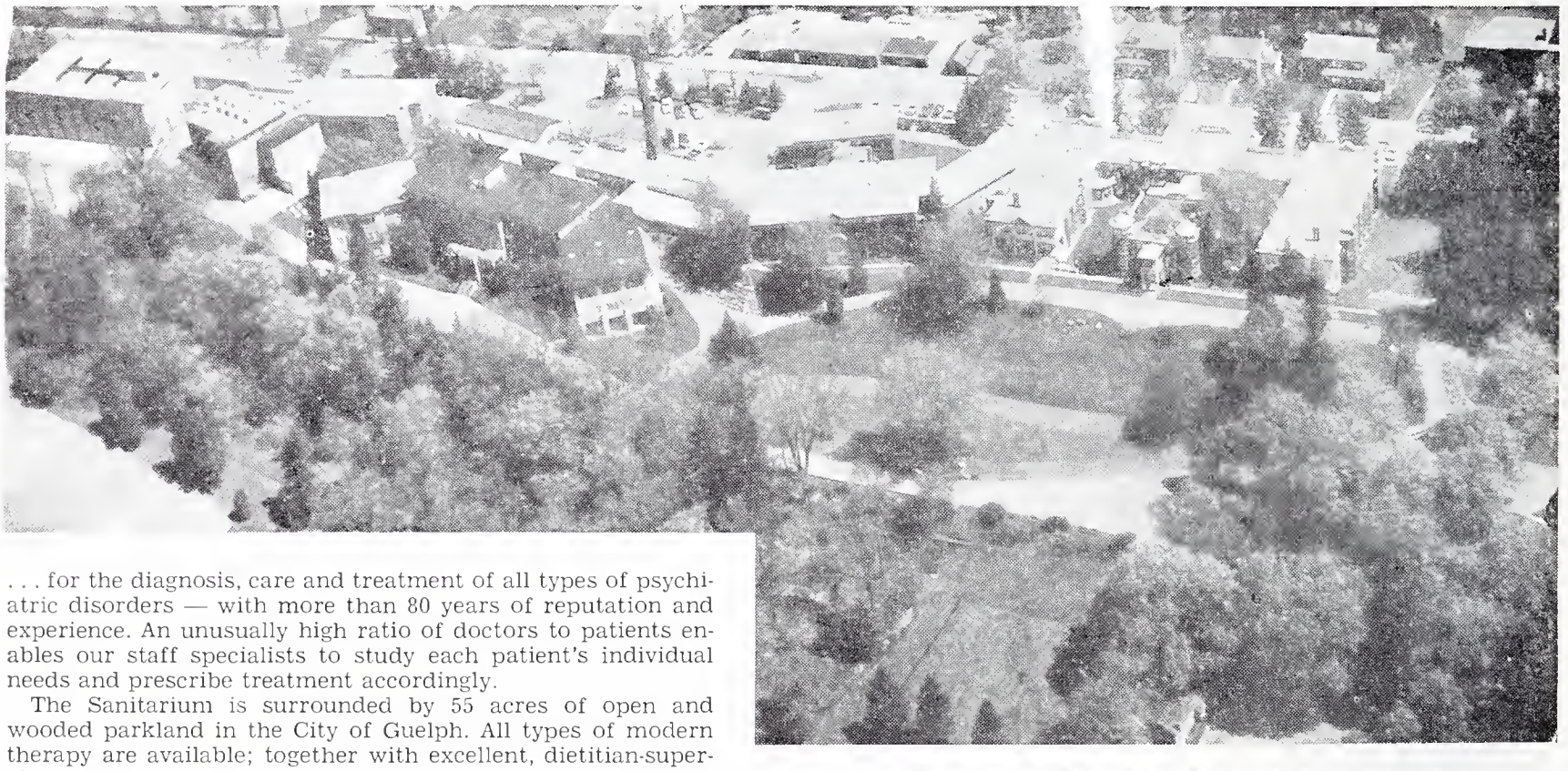

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